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CASE REPORT

Acute spinal cord dysfunction in bacterial meningitis: MRI findings

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Introduction

Acute spinal cord dysfunction is a rare complication of bacterial meningitis; approximately 33 cases have been reported in the English literature, mostly before the advent of magnetic resonance imaging (MRI).¹ Spinal MRI during the acute stage of spinal cord dysfunction has so far been reported in only three children and five adults.¹⁻⁶ We report the MRI findings of an adult female patient who developed spinal cord dysfunction during the acute stage of bacterial meningitis.

Case report

A 37-year-old woman was admitted to hospital complaining of fever and weakness for 3 days. Physical examination revealed tonsillitis, and intra-muscular penicillin (single dose of 1.2 million units of benzathin penicillin and twice daily 800,000 units of penicillin G for 5 days) was administered without previous cultures. Baseline laboratory investigations showed a white blood cell count of $20.2 \times 10^9/l$ (normal range: $4.8-10.8 \times 10^9/l$) with 90% neutrophils; an erythrocyte sedimentation rate of 100 mm/h and C-reactive protein level of 210 mg/l (normal range: 0-6 mg/l). Shortly after admission she complained of headache, at which time her body temperature was 39 °C. Examination disclosed neck stiffness and positive Kerning and Brudzinski signs. Cranial MRI was normal (not shown). Meningitis was suspected and spinal tap was performed. Cerebrospinal fluid (CSF) showed glucose of 1.77 mmol/l (less than 40% of the serum glucose level), protein of 2230 mg/l and 2000 cells per mm³ with 80% neutrophils. Gram stain of the CSF and cultures of blood were unremarkable, and CSF culture did not grow any bacteria. The patient was treated with intravenous antibiotic therapy (vancomycin: 2 g/day, cefepime: 4 g/day) and intravenous methylprednisolone (40 mg/day). The following day, bladder incontinence and paraplegia developed (muscle tone

power grade 0/5 in the legs). Muscle tone and tendon reflexes were mildly decreased in the legs compared with the arms with absent abdominal skin reflexes. No clear sensory level could be determined, but vibratory sense was decreased in both legs. Spinal MRI demonstrated contiguous hyperintensities on T2-weighted images extending into the whole thoracic spinal cord. Swelling of the spinal cord and leptomeningeal enhancement were observed on T1-weighted images (Figs. 1 and 2). Five days later, she was afebrile and all symptoms except bladder incontinence and paraplegia had resolved. Four weeks after the onset of meningitis, follow-up examination showed paraplegia (2/5 bilaterally) of lower extremities without sensory loss or incontinence. Eight months later, mild spasticity was observed. On follow-up MRI, the intra-medullary signal abnormalities were completely resolved (Fig. 3).

Discussion

Bacterial meningitis is frequently accompanied by intra-cranial or systemic complications.⁷ Spinal involvement is rare in non-tuberculous bacterial meningitis. Most of the cases have been reported in children.¹ The frequent causative pathogens are *Neisseria meningitidis*, *Streptococcus pneumoniae*, *Haemophilus influenzae*.¹

Cervical lesions predominate in young children and infants in contrast to the preponderance of midthoracic and lumbar cord lesions in adolescents. The reasons for this apparent age-related difference are not known.⁸ The time of onset of spinal cord symptoms varies from the time of diagnosis of meningitis to 4 days after initiation of treatment of the infection. The most common initial symptom is quadriplegia or paraplegia.¹

Meningitis-related myelopathy is an unfavourable prognostic indicator. Most patients have poor recovery of cord function. Moffett and Berkowitz¹ reviewed 23 patients with spinal cord dysfunction after bacterial meningitis. Of the 18 survivors, only two demonstrated complete neurological recovery, and the remaining 16 patients exhibited spastic

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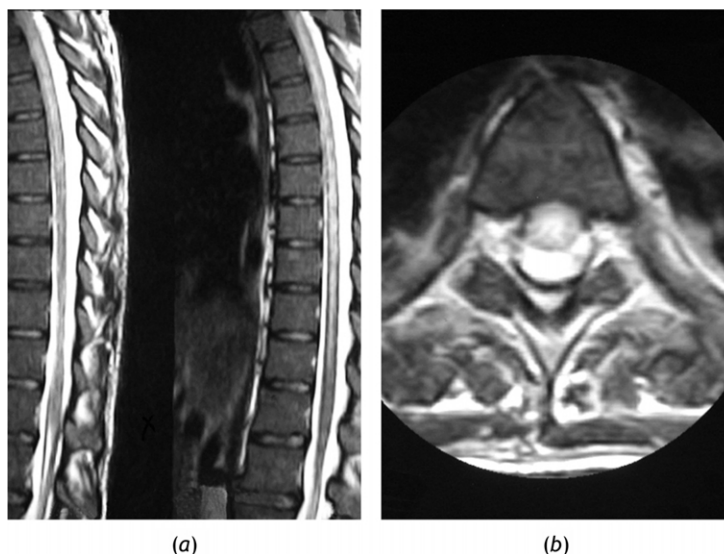


Figure 1 (a) Sagittal T2-weighted MR images show hyperintense lesions involving the whole thoracic spinal cord. (b) Axial T2-weighted MR image at mid-thoracic level shows hyperintense lesions involving the central spinal cord.

quadriplegia, paraplegia, with increased deep tendon reflexes, walking difficulties, and abnormalities of bowel and bladder functions.

The pathogenesis of spinal cord dysfunction complicating bacterial meningitis may involve several mechanisms. Although a surgically treatable empyema or abscess within or contiguous with the spinal canal should be considered when signs of cord dysfunction develop, such lesions are not found in patients in whom myelography or MRI was performed.^{4,8} Impaired blood flow and cord ischaemia are the probable causes of dysfunction in the majority of patients. Microscopic studies have shown endothelial cell swelling, vasculitis and

venous thrombosis of subarachnoid vessels during the acute phase of meningitis; and these changes may lead to vessel occlusion and parenchyma infarction. Vasculitis, particularly arteritis, plays an important role in the neurological damage occurring in acute bacterial meningitis.¹ Other causes of vascular compromise to the spinal cord include hypoperfusion resulting from cardiorespiratory arrest and hypotension. An infarction involving the anterior spinal artery would be expected to involve the spinal cord bilaterally with variable contrast enhancement and should result in tissue loss.⁹

Spinal cord involvement may be due to myelitis

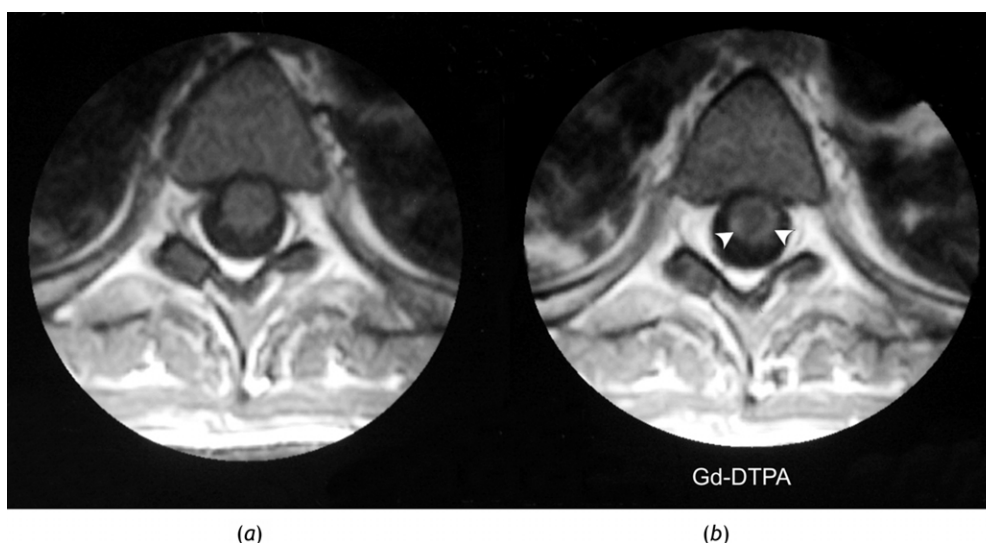


Figure 2 (a) Pre-contrast and (b) post-contrast axial T1-weighted images shows leptomeningeal enhancement (arrows) in mid-thoracic level.



Figure 3 On follow-up sagittal T2-weighted MR images show no pathological signal in thoracic spinal cord.

in acute phases of bacterial meningitis.^{4,5} Myelitis during purulent meningitis has been demonstrated on post-mortem examination. Oedema, focal haemorrhages, perivascular inflammation in the subarachnoid space, capillary thrombosis, thrombosis of anterior spinal artery, and myelomalacia has been reported. Myelomalacia primarily affected the grey matter while the white matter was spared.

The spinal MRI findings during the acute stage of spinal cord dysfunction due to bacterial meningitis have been reported in few cases. These cases are summarized in [Table 1](#). The findings in meningitis-related myelitis in the previously published cases^{4,5} included contiguous intra-medullary hyperintensities on T2-weighted images extending over several spinal cord segments with or without spinal cord swelling. Medullary, leptomeningeal enhancement may be seen on T1-weighted images. These findings are congruent with ours.

Our patient did not experience hypotension or shock and the resolution of the extensive intramedullary signal abnormalities can not be attributed to ischaemia. Moreover, the spinal cord lesions were multilevel and did not follow an arterial distribution. The MRI findings in our case were

Table 1 Clinical/MRI features of eight patients with acute spinal cord dysfunction complicating bacterial meningitis

Ref.	Age/sex	Organism	Time of onset	Location	Initial sign	T1W	T2W	Enhancement	Follow-up MRI	Possible mechanism	Outcome
2	15m/M	<i>H. influenzae</i>	1 day	Craniocervical	Tetraplegia	Hyperintense	Hyperintense	Medulla	*	Vasculitis	*
6	25y/F	<i>N. meningitidis</i>	3 day	Lower medulla, upper cervical	Tetraplegia	*	Hyperintense	*	*	Infarct	Died
5	17y/M	<i>N. meningitidis</i>	4 day	Thoracic (T4- T9)	Paraplegia	Isointense	Hyperintense	None	*	Vasculitis, myelitis	Paraplegia
4	36y/F	<i>S. pneumoniae</i>	same day	Cervical to lumbar	Tetraplegia	Hypointense	Hyperintense	Medulla, leptomeningeal	Resolved and newly formed syrinx	Myelitis	Tetraparesis, bladder, bowel incontinence
4	33y/M	Unknown	same day	Cervical to lumbar	Paraplegia, bladder, bowel incontinence	Isointense	Hyperintense	None	Resolved	Myelitis	Bladder, bowel incontinence
4	17y/M	<i>N. meningitidis</i>	1 day	Cervical to thoracic	Respiratory arrest	Isointense	Hyperintense	None	Progressed	Myelitis	Tetraparesis, bladder, bowel incontinence
3	14m/F	<i>S. pneumoniae</i>	initial presentation	Cauda equina	Flaccid paraplegia	Isointense	*	Cauda equina, dural sac	Reduced enhancement	*	Bladder, bowel dysfunction
1	4d/F	<i>E. coli</i>	1 day		Respiratory distress	No abnormalities	No abnormalities	*	*	Vasculitis	

T1W, T1-weighted MR images; T2W, T2-weighted MR images. Enhancement, after intravenous Gd-DTPA injection. *Undefined.

compatible with myelitis or vasculitis. However, high signal on T2-weighted images in the central spinal cord is a common finding in venous congestion, which might have contributed to the spinal cord damage.¹⁰

On follow-up, the signal abnormalities seen on MRI resolved completely in one reported patient, as in the present case, however, another patient was left a small syrinx in the cervical cord, and other cases have been reported to progress.⁴

In conclusion, this case illustrates that myelopathy may complicate bacterial meningitis. Despite a normal follow-up spinal MRI, neurological deficits should be attributed to damage to the spinal cord.

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